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CLINICAL PROCEEDINGS

OF THE CHILDRENS HOSPITAL

13th and W Streets, Washington 9, D. C.

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POLIOMYELITIS SURVEY FOR 1951

Special Report No. 239

David L. Simon, M.D.
Joseph M. LoPresti, M.D.

This study represents a survey of the poliomyelitis patients admitted to the Children's Hospital of Washington, D. C. during the period from January 1 to December 31, 1951. During the past endemic season, acute anterior poliomyelitis laid a gentle hand on the children of the District of Columbia. In the 12-month interval studied, only one hundred and thirty-three pediatric patients were treated for poliomyelitis. In all instances, a definite diagnosis was established by a correlation of the history, physical findings, and spinal fluid changes.

The mildness of the disease during the period surveyed is illustrated in Table 1. Seventeen or 12.5 per cent of the children had a significant degree of paralysis. There were five patients who had bulbar involvement; and one child, classified as bulbospinal in type, had to be placed in a respirator. No fatalities occurred in this group of children.

The sex and race distribution demonstrates some interesting facts (Table 2). There were 114 white patients and 19 colored ones, a ratio of 5 to 1. This figure is significant and lends itself to some speculation. It has been noted in the past that acute anterior poliomyelitis occurs with greater frequency in children from the higher economic groups. A possible explanation for this phenomenon might lie in a number of factors. In the lower economic classes sanitation and personal hygiene are neglected. It could be assumed, therefore, that the opportunity for this group of children to come in contact with poliomyelitis virus is enhanced. The logical likelihood, then, is that they acquire an early immunity to the disease. A fact more difficult to explain is why the disease was twice as common in males than in females.

The age distribution is shown in Table 3. The youngest patient was seven months old, and the oldest was 14 years of age. Twenty-six or 19.5 per cent of these children were between 5 and 6 years. The majority (87 or 65 per cent) of the cases occurred between the ages of 2 and 8. Poliomyelitis is a rare disease in infancy and only eleven of the patients were under 2.

The fact that acute anterior poliomyelitis is a seasonal disease, occurring endemically and epidemically during the enteric and fly seasons, is well-established (Table 4). This is borne out by our study. One hundred and twenty-five or 94 per cent of the cases were hospitalized during the 5 month period from June through October. Cases of poliomyelitis may occur sporadically throughout the year and in this survey January was the only

TABLE 1

Classification

TYPE	NUMBER	PER CENT
Non-paralytic	97	72.9
Paralytic	30	22.9
Severe	17	12.5
Mild	13	10.4
Bulbar	5	3.8
Bulbo-spinal	1	0.7
Total	133	100

TABLE 2

Sex and Race Distribution

	WHITE	COLORED	TOTAL
Male	77	13	90
Female	37	6	43
Total	114	19	133

TABLE 3

Age Distribution

AGE (YRS.)	NUMBER	AGE (YRS.)	NUMBER
0-1	2	7-8	14
1-2	9	8-9	5
2-3	12	9-10	15
3-4	10	10-11	4
4-5	12	11-12	6
5-6	26	12-13	3
6-7	13	13-14	2
Total	84		49

TABLE 4

Month of Distribution

MONTH	NO.	MONTH	NO.
Jan.	0	Jul.	17
Feb.	1	Aug.	49
Mar.	1	Sep.	37
Apr.	1	Oct.	11
May	1	Nov.	3
Jun.	11	Dec.	1
Total	15		118

month during which no child was admitted with this diagnosis. The greatest number of children were admitted during August and September (49 and 37 respectively).

Most authorities agree that this disease is infectious and contagious (Table 5). A definite history of contact was elicited in only ten (13 per cent) of the cases studied. In two instances in this series, one of the parents of a child with poliomyelitis was admitted elsewhere with the same disease.

Russel in England first observed that exertion in the early stages of acute anterior poliomyelitis increased the severity and degree of paralysis. This relationship of exertion to muscular involvement now is unquestioned (Table 6). Our findings in this respect are not significant. Fourteen children engaged in rather strenuous exertion in the early stages of their illness. Of these, four had an ensuing paralysis.

TABLE 5

Infectious Nature

Contact History	10 patients
Siblings	3 instances
Parents	2 instances
Total	15

TABLE 6

Exertion

History of exertion	14
Non-paralytic	10
Paralytic	4
Total	14

Reports from England and Australia and, recently, from the United States indicate a relationship between injections and an increased incidence of paralysis. It would appear also that the localization of paralysis is influenced by the site of injections. This relationship apparently was substantiated by two cases in our survey. The first was a seven-month old white male who received a DPT inoculation in the right triceps two weeks before admission; on examination, there was a paralysis of the right upper extremity present. The second was a three year old white male who was given a penicillin injection into the left buttock one week prior to hospitalization. At the time of admission, weakness of the left leg was noted. However, it should be pointed out that a total of 42 patients received an injection (penicillin, tetanus, or adrenalin) shortly before or during their illnesses.

A prodromal phase is frequently encountered in poliomyelitis. In 32 of the children studied (24 per cent), the onset of the illness was heralded by a short, two to three days, prodrome of non-specific symptoms, e.g., slight fever, malaise, rhinitis and sore throat. Two of these children were noteworthy and serve as precautionary examples to the medical practitioner. A seven-year old white male, whose sibling was hospitalized with a diagnosis of bulbar poliomyelitis, was admitted with minimal symptomatology. His spinal fluid examination was normal. Four days later, this patient developed more specific symptoms, and characteristic spinal fluid changes. The second case was a nine-year old white female whose younger sister was in a respirator. The symptoms again were non-specific and after a normal spinal fluid examination, she was sent home. Five days later, this child was re-admitted

TABLE 7

Symptoms

	NUMBER	PER CENT
Fever	133	100
Headache	97	73
Vomiting	86	65
Stiff neck	63	47
Lethargy	57	43
Stiff back	53	40
Sore throat	44	33
Nausea	43	32
Muscle pain or spasm	28	21
Constipation	27	20
Delusions	17	13
Abdominal pain	14	11
Weakness or paralysis	13	10
Diarrhea	13	10

to the hospital with classical physical findings. At this time, spinal fluid changes were present. Both of these cases serve to illustrate two facts; viz., (1) the spinal fluid early in the course of acute anterior poliomyelitis may be normal, and (2) the finding of a normal spinal fluid does not release the physician from the responsibility of keeping suspected cases of poliomyelitis at bed rest for a safe period of time.

The usual child with poliomyelitis presents a fairly definitive and easily recognizable clinical picture. He is, most often, a five or six year old white male who has been ill for five to six days before admission (average 5.3 days (Table 7). Fever is invariably present; it will not be high and will range between 101 and 103 degrees. A severe, non-localized headache is often a major complaint. Vomiting, frequently projectile in nature and

usually repeated, will have occurred. A stiff neck and back are common manifestations and a history of sore throat often is present. One of the most usual complaints is that the child has become lethargic. While abdominal pain is not a common feature, it may be present and on occasion may be a source of confusion. The combination of fever, vomiting, and pain in the abdomen suggests a surgical problem. Indeed, one child in this series, a three year old colored male, was suspected of having acute appendicitis. He was observed for 24 hours before a diagnosis of poliomyelitis was established.

On physical examination, the neurological nature of the disease is apparent (Table 8). The patient has a flushed facies with circumoral pallor. Nuchal rigidity is the most common finding. Back spasm is usually present,

TABLE 8

Physical Findings

	NUMBER	PER CENT
Nuchal rigidity	120	90
Back spasm	62	47
Dec. deep reflexes	57	43
Hamstring spasm	54	41
Dec. superficial reflexes	53	40
Flushed facies	53	40
Positive Kernig's	40	30
Weakness or paralysis	36	27
Inflamed throat	20	15
Positive Brudzinski's	18	14
Positive Babinski's	17	13
Abdominal pain	13	10
Increased reflexes	9	7

and on sitting, the child assumes the tripod position. The superficial and deep reflexes commonly are found to be depressed or absent. Straight leg raising is limited because of bilateral hamstring spasm. A frequent finding is the presence of a positive Kernig's sign.

The spinal fluid examination often is not essential to establish the diagnosis of acute anterior poliomyelitis. However, it must be performed in all cases to differentiate other central nervous system diseases (Table 9), particularly bacterial meningitis. In poliomyelitis, the spinal fluid is clear or slightly hazy. There is a mild increase in the cellular content, rarely over 300 cells per cubic millimeter. Early, most of these cells are polymorphonuclear leukocytes; in the second week, there is a shift and most of the cells are lymphocytes. The protein content is moderately increased and this change may persist for a month or more. Since the protein elevation fre-

quently outlasts the pleocytosis in the spinal fluid, the possibility presents itself that patients who are subjected to spinal puncture late in the course of their illness, may be confused with the Guillain-Barre syndrome. The sugar and chloride levels are unaffected.

Four patients were admitted to the isolation ward who later were shown to have a different disease entity which at first simulated poliomyelitis. In two instances, rheumatic fever proved to be the imitator. The pseudo-paralysis and muscular tenderness attending some cases of acute rheumatic fever serve to becloud the issue. However, the normal spinal fluid together with joint involvement and an elevated erythrocyte sedimentation rate

TABLE 9

Spinal Fluid (Average)

Appearance:	Clear or slightly hazy
Cells:	113 WBC's/cu. mm.
	Polys 55 per cent
	Lymphs 45 per cent
Protein:	38 mgms. per cent
Sugar:	48 mgms. per cent

TABLE 10

Paralysis or Weakness

LOCATION	NUMBER
Lower extremity	14
Upper extremity	10
Cranial nerve	5
Neck	4
Back	2
Intercostal	1
Total	36

soon established the correct diagnosis. In one child, an hemophilus influenzae meningitis was the etiologic factor and illustrates the necessity for spinal fluid examination in all suspected cases. Of particular interest was a four year old white male who had a specific enteritis caused by the shigella group of organisms. Central nervous system manifestations accompanying shigellosis are a well-documented fact. However, our patient was unusual in that a pleocytosis was present in his spinal fluid.

The majority of patients had no weakness or paralysis and were permitted to go home at the end of their isolation periods (two weeks from the onset of symptoms) (Table 10). Thirty-six or 27.1 per cent of the chil-

dren had muscular weakness or paralysis. Of these, seventeen (12.5 per cent) require braces or supports. Of the ninety-seven patients who were examined and treated by the Department of Physiotherapy, sixty-seven did not need any treatment on discharge from the hospital. One patient with rather extensive involvement is still hospitalized.

Two principles were followed in the management of these patients in the acute stage of the illness, viz. (1) to avoid exertion, and (2) the prevention of deformities. The most important feature was absolute bed rest; the patients were not permitted even to feed themselves. They were allowed to assume any position which was restful. If positioning of parts did not interfere with the child's rest, then neutral anatomic positioning was attempted. The lay-on type of hot pack was utilized sparingly in this stage of the illness, and only when pain and spasm were so severe that they interfered with the patient's rest. Hot packs were ordered for half-hour periods three to four times a day. Sedation with the opiates was instituted to control pain or spasm only if bulbar or respiratory involvement were absent. An extra salt intake in the form of thermotabs, one-half to one tablet three times a day, was given when hot packs were utilized. The diet was a high-caloric, high-protein one; and vitamins were administered to all patients. Whenever it was feasible, early and gentle passive physiotherapy was instituted. In a significant number of patients (nine or 7 per cent) urinary retention presented a problem. This was efficaciously combatted in all instances by the parenteral use of furmethide, in a dose of ¼ to 1 cubic centimeter given subcutaneously.

SUMMARY

One hundred and thirty-three children with poliomyelitis were admitted to Children's Hospital, Washington, D. C. in the 12-month period from January 1 to December 31, 1951.

There were no fatalities. Ninety-seven or 72.9 per cent were nonparalytic; seventeen or 12.5 per cent had significant paralysis; there were five bulbar patients, and one child had to be placed in a respirator because of intercostal involvement.

One hundred and fourteen of the children were white and 19 were colored (a ratio of 5 to 1). Males outnumbered the females 2 to 1.

The majority of cases (87 or 65 per cent) occurred in children between the ages of two and eight. One hundred and twenty-five of these children (94 per cent) were admitted in the five-month period between June and October, the fastigium occurring during August and September.

The infectious nature of poliomyelitis and the relationship of exertion and inoculations to paralysis were demonstrated in an insignificant number of the children studied.

An easily recognizable symptomatology of fever, headache, vomiting, stiff neck and back, and lethargy was present in the majority of instances. On physical examination, the most usual findings were nuchal rigidity, back and hamstring, spasm, and decreased superficial and deep reflexes.

Spinal fluid findings consisted of a mild pleocytosis and moderate increase in protein content.

Rheumatic fever, bacterial meningitis, and specific enteritis (shigellosis) simulated poliomyelitis.

Thirty-six or 27.1 per cent of these children had muscular weakness or paralysis; seventeen or 12.5 per cent now need braces or supports.

The plan of management in the acute stage was to avoid exertion and prevent deformities.

AN UNUSUAL RACHITIC DEFORMITY CORRECTED BY WEDGE CASTING

Case Report No. 240

Alfred C. Nowak, M.D.
Francis M. Mastrota, M.D.

Rickets is still somewhat common today in mild forms, but the severe deformities do not occur so frequently. This is probably due to an increased knowledge of nutrition and the widespread use of potent vitamin D supplements. Except in urban areas and in the colder climates, the short wave ultra-violet of the sun may still exert some anti-rachitic effect. The following case is an example of severe rachitic deformity which was treated with combined orthopedic, medical, and psychiatric care.

CASE REPORT

W. M., a 30-month old colored male was presented at the orthopedic clinic by his mother who had noted that his "knees bent the wrong way." He started to walk at 12 months of age but no deformity of the legs was apparent until 5 months later. In the following year the waddling gait and the deformity became more severe. A detailed history, obtained by the resident and a psychiatric social worker, revealed that the patient refused to drink any milk after he was weaned from the breast at nine months. His mother did, however, offer him some ice cream almost every day to make up the milk deficit. He was maintained on Vi Synerol® for the first 12 months but after this time he received no vitamin supplements. Except for the rejection of milk and egg yolk the remainder of his dietary history was normal and he ate all foods eagerly. Physical examination revealed a 31-pound colored male who was in no distress. The anterior fontanelle was patent about 2 centimeters. The teeth were well formed for his age. The abdomen was slightly protuberant and he had flaring of the wrists and

costrochondral junctions. His knees were greatly angulated with the right knee being angulated laterally and the left angulated medially (Fig. 1).

The significant blood chemistries on admission were as follows: calcium, 9.6 milligrams per 100 milliliters; phosphorous, 2.4 with a calcium phosphorous product of 23.04. The phosphatase was 33.0 B.U. Urinalysis and hemogram were normal. Electrocardiogram showed a rate of 160 with no changes of the T wave or QT interval. X-ray of the lower extremities (Fig. 3) revealed the rachitic changes, as did the x-rays of the chest and upper extremities.

Treatment was instituted after the basal studies were completed. A double hip spica was applied at the same time correcting the deformities as much as possible. Ten days later, the cast was wedged at the knees and further corrections were made

FIG. 1. Before casting

FIG. 2. After casting

aligning the lower legs with the upper. X-rays taken after the wedging revealed the bones to be in good alignment. From the onset of his hospital course he was offered an adequate diet and he accepted milk eagerly when it was flavored and colored with chocolate syrup. The psychiatry resident and a psychiatric social worker visited him daily. Following the orthopedic corrections, the active medical treatment was instituted after doing further blood chemistries, which revealed the alkaline phosphatase to be 36.0 B.U., calcium 9.4 mgms.%, phosphorous 1.3 mgms.% with a calcium-phosphorous product of 12.22. The patient received 5,000 units of vitamin D every day along with three grams of calcium gluconate. Ten days later, x-rays of the wrists showed response to the anti-rachitic therapy. The patient was discharged to home care with the spica in place and continued vitamin D and calcium gluconate therapy.

Thirty days later, the cast was removed and the legs were noted to be straight. Weight bearing was restricted and graded physiotherapy was started to restore muscle tone and strengthen the ligaments.

FIG. 3. Before casting

FIG. 4. After casting

DISCUSSION

When rickets occurs in a child there is a disturbance of the calcium and phosphorous metabolism preventing the normal deposition of calcium salts in the growing parts of the skeleton. The skeleton becomes weak and being unable to stand the stress to which it is ordinarily subjected, it must yield and become deformed. The constant disintegration of old lime-containing bone and the production of bone devoid of lime salts causes the skeleton to become so weak that it bends under the weight of the body and the pull of its muscles. The bending of the long bones most often has its origin at the junction regions of the epiphysis and diaphasis. The minor symptoms of rickets such as sweating, irritability, restlessness and digestive disturbances are insidious in their onset. Generally the first signs are skeletal changes. If the infant is about three months old, craniotabes may be the first and only sign of the disease. When rickets is allowed to continue the craniotabes disappears and thickening of the central part of the frontal and parietal bones produces the characteristic natiform or "hot cross bun" head. Other rachitic deformities are produced largely by the time in the child's life in which the disease is active. The changes of stress on the bones varies with the changing demands of posture and activity. During the period of infancy,

when the infant is recumbent, the skeletal changes are limited to the head, thorax and pelvis and to a small extent to the extremities. When the child begins to sit up, the direction of gravity is changed, tending to produce dorsal kyphosis.

Deformities of the lower extremities often form before the infant sits up. He usually has one leg crossed over the other to maintain his balance and the bones become bent by the force of gravity and the pull of the shortened flexors. When a rachitic child starts to stand and walk the type of deformity depends somewhat on the deformity that already exists in the pelvis, femora and tibiae. The variety of deformities include bowing, knock knee and combinations of both. During the second and third year the growth of the long bones is most rapid and severe angular deformities occur at this time.

Multiple fractures in rachitic infants and children can add to the complexity of deformities. Fractures usually occur on one side of the cortex with the other side merely bending. These fractures may be discovered by x-ray when otherwise they are unsuspected. Usually no deformity results until the fracture begins to heal. After the fourth year rickets more closely resembles the osteomalacia of the older child and it progresses with bending deformities. Curvatures of the shaft may be the result of bending fractures or tilting of the epiphysis on the diaphasis. Deformities resulting from comcomplete fractures do not conform to any type. Those caused by tilting of the epiphysis and bending of the shaft conform to certain types depending upon the age the rickets occurs and the stress of the postural habit. Asymmetrical bowing may occur when the child spends a good part of the day sitting. Knock knees may be due to medial angulation of the knees or medial bowing of the femora and tibiae. It is not always symmetrical and it is frequently associated with scoliosis and coxa vera. Relaxation of the knee joints with poor muscle tone is a frequent cause of knock knees in nonrachitic children.

Coxa vera or a downward deflection of the neck of the femur is another produced by severe rickets. It is usually associated with other deformities and it is almost always bilateral.

CONCLUSION

This case presented is an example of a severe rachitic deformity successfully treated by the combined efforts of the orthopod, the pediatrician and the social worker.

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HYPOPITUITARISM IN INFANCY

A Report of a Case Occurring in One of Twin Girls

Case Report No. 240

Joseph M. LoPresti, M.D.
David A. St. Martin, M.D.
Reginaldo Pascual, M.D.

INTRODUCTION

The following case history is deemed worthy of reporting because it presents the opportunity to discuss briefly the problem of abnormal growth patterns in childhood.

CASE REPORT

This twenty-one month old white female was admitted to the Children's Hospital, Washington, D. C. with the chief complaints of growth failure and fever.

The patient was the second born of twins, the first being born two to three minutes ahead of her. They were full term babies of normal, uneventful deliveries. The first three months of the mother's pregnancy were characterized by nausea, vomiting, anorexia, headaches, and dizziness. However, at the start of the second trimester, these symptoms disappeared spontaneously, and the gestation progressed normally. The patient's birth weight was 2 pounds, 14 ounces, while the twin weighed 5 pounds, 5 ounces. The premature infant was hospitalized for four months after birth during which her weight gain was retarded markedly, approximately 2 ounces per week. During this period intermittent oxygen therapy was necessary and several febrile episodes of undetermined origin were observed. She was discharged from the hospital at 4 months of age and weighed 5 pounds. At home, the feeding history was adequate, however, growth retardation persisted. In spite of numerous formula changes and the addition of solid foods, the infant weighed only 8 pounds, 8 ounces at the age of 17 months. During this interval a diagnosis of hydrocephalus was suspected but encephalography failed to confirm this suspicion. Occasional febrile episodes were noted but no etiology could be indicted.

There was no history of vomiting, diarrhea, cough, difficulty in breathing, or convulsions. The first tooth erupted at 12 months of age; the vocabulary is limited to a few, single words; and she has never been able to sit up. The patient was able to hold her head up at the age of 17 months.

The father and mother are normal and there is no consanguinity. Of three siblings, one died in infancy of pneumonia; the twin and older sibling are normal. A family history of tuberculosis was elicited on the maternal side, but no abnormalities of growth or stature are present in either branch of the family tree.

On physical examination, the general appearance was that of a symmetrically

---

N. B. This patient was under the private care of Morris Michael, M.D. and was admitted to the ward service of Preston A. McLendon, M.D.

stunted infant. The following chart compares the bodily measurements of the patient, her twin, and the normal average (cf. Figs. 1 and 2):

MEASUREMENT	PATIENT	TWIN	NORMAL
Weight	3.08 kgm.	8.18 kgm.	11.1 kgm.
Height	55 cm.	71 cm.	81.9 cm.
Head circumference	43 cm.	47 cm.	47.9 cm.
Chest	35 cm.	44 cm.	38.6 cm.
Abdomen	31 cm.	38.5 cm.	48.5 cm.

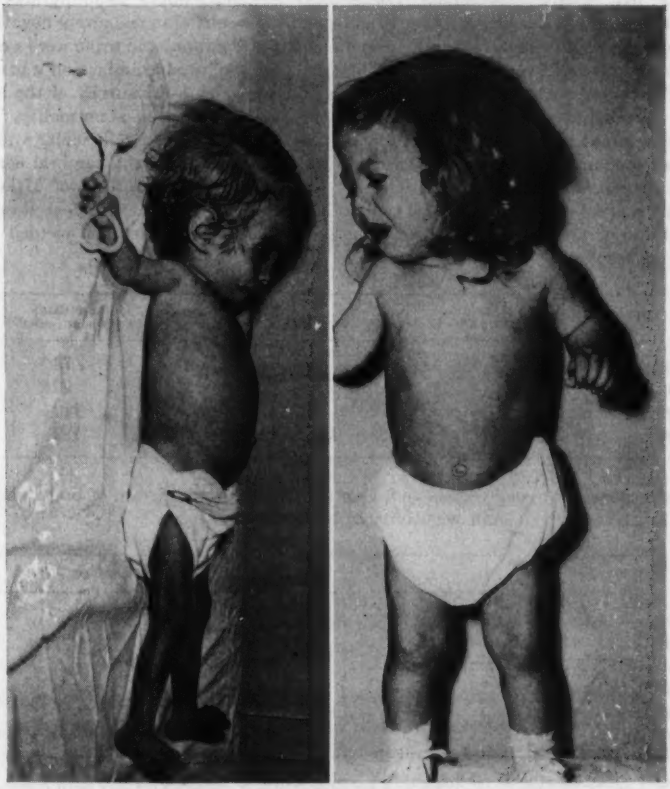

FIG. 1

The head was brachycephalic with a cephalic index of 85. The anterior fontanelle was patent and measured 2.0 centimeters. The calvarium was prominent and mild frontal bossing was present. The hair was normal. The nose was saddle-shaped and

small. Only 12 teeth had erupted. The neck was short. A pigeon-breast was noted. The extremities were small, but in proportion to the trunk. The skin was pale and yellow-tinged. The remaining physical findings, including the neurological examination, were normal.

An intensive laboratory examination was instituted. The hemogram and urinalysis were essentially normal. The hematocrit was 42. The blood cholesterol was 182 milligrams per 100 milliliters. The blood potassium, sodium, calcium, and phosphorous levels were within normal limits. The non-protein nitrogen was 34 milligrams per 100 milliliters; the BUN, 24; and the carbon dioxide combining power, 41 volumes per 100 milliliters. Liver function tests were normal. A fasting blood sugar was 100 milligrams per 100 milliliters and the glucose tolerance test yielded the following results: fasting, 100; ½ hour, 128; 1 hour, 128; 2 hours, 75; and 3 hours, 78 milligrams per 100 milliliters. The serologic test for syphilis and tuberculin skin tests were negative. Trypsin was present in the stool in normal amounts. The fundi and urine were examined for cystine crystals and none were found. The spinal fluid examination was normal. Phenylpyruvic acid was not present in the urine. An x-ray survey of the long bones revealed marked delay in ossification centers but no other abnormality. The skull was infantile and pneumoencephalography showed a normal ventricular system with an accumulation of an excessive amount of air over the right cerebral cortex which suggested focal cortical atrophy. The total daily urinary output of 17 ketosteroids was less than 1 milligram per 100 milliliters. The effects of a test dose of ACTH and of adrenalin on the circulating eosinophils were studied and yielded the following results:

TIME	ACTH (EOS./CU. MM.)	ADRENALIN (EOS/CU. MM.)
Fasting	88	73
1 hour	60	90
2 hours	53	110
3 hours	0	182

The response of hypoglycemia to 0.1 unit of insulin per kilogram of body weight given intravenously (insulin sensitivity test) showed:

TIME	BLOOD SUGAR MGM/100 ML
Fasting	80
½ hour	45
1 hour	40
1½ hours	50
2 hours	50
3 hours	60

DISCUSSION

The pediatric age group affords the practitioner an unique opportunity to study the effects of endocrine imbalances. While in the adult, endocrine dysfunction is usually attended only by metabolic disturbances; in the child

an endocrine imbalance may alter seriously the rate of osseous maturation, body growth, or the development of secondary sex characteristics. At times the effects on growth may be so marked that an impression may be gained from a simple clinical inspection of the patient, e.g., the child with cretinism. It should be stressed that the growth pattern in childhood may be altered by a number of factors unrelated to dysfunction of the glands of internal secretion. The inclination to label any disturbance in growth as "gland trouble" is a common pitfall. Whenever an endocrine disturbance is sus-

FIG. 2

pected as the cause of an abnormal growth pattern, then physiological and chemical studies must be performed to establish the diagnosis.

Numerous extrinsic and intrinsic factors may alter the pattern of human growth and development. Figure 3 is an attempt to schematically represent the interrelationship of these numerous influences.

Extrinsic factors and environment may affect profoundly the growth pattern in childhood and may be responsible for marked deviations from the normal:

1. *Nutritional factors:* The quality of food and the caloric, vitamin, and mineral intake exert important influences on growth. Not only is this true, but it is also essential that digestion, absorption, circulation, oxygen saturation of the blood, and assimilation be unimpaired for normal growth to occur.

2. *Maternal factors:* The toxemias of pregnancy or other diseases in the mother may play an important role in the ultimate constitutional make-up of the individual. The baneful effect of rubella in the first trimester of pregnancy is a well-substantiated medical fact.

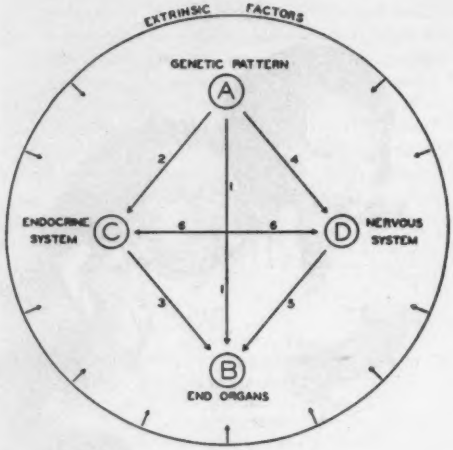

FIG. 3

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3. *Intrauterine factors:* Placental hemorrhage, impaired fetal circulation, and fetal anoxia may influence future growth and development.

4. *Chronic infection:* Chronic disease of the kidneys, pancreas, or liver at any time during intra- or extrauterine life may alter the rate and pattern of growth.

The intrinsic influences play as important a role as do the extrinsic ones. The genetic factors with which the zygote is endowed in utero determine, to a large extent, the constitutional pattern of the end organ. In the early fetus, a "genic hormone" is circulated which influences the type and degree of cellular division and specialization. Later in fetal life, as the organism becomes more differentiated some of these genetic influences on cellular growth are relegated to the specialized organs of internal secretion. Endocrine function, in turn, is regulated by neural impulses. At any time genic

mutations and alterations may be produced by extrinsic environmental factors (cf. Fig. 3).

1. The basic genetic pattern may directly influence the growth and development of the individual. This is indicated by pathway A–B in Figure 3. Examples of this type of genetic influence may be found in acrondroplasia, hypertelorism, mongolism, Laurence-Moon-Biedl syndrome, tendency to obesity or leanness.

2. Genetic factors may produce constitutional differences in the endocrine glands which, in turn, cause devious growth patterns in the end organ as indicated by pathway A–B–D. Such a sequence probably occurs in cases of thyrotoxicosis, certinism, and diabetes mellitus.

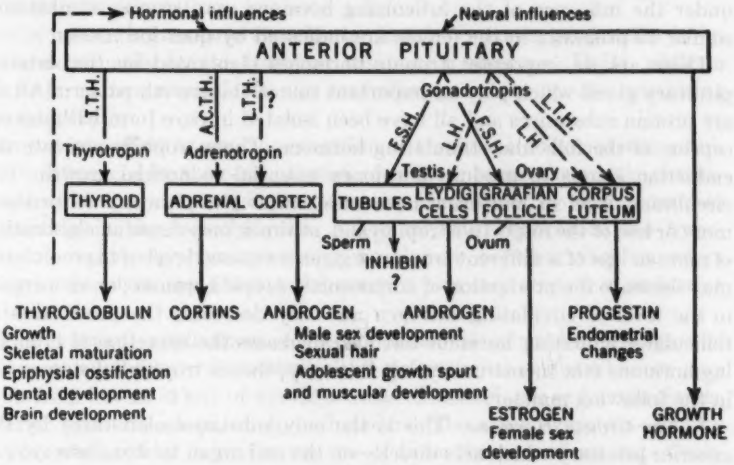

FIG. 4

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3. Genetic influences may cause alterations in the neural impulses which activate endocrine function as illustrated by pathway A–C–B–D. Examples may be found in some cases of Cushing's syndrome, sexual precocity, and sexual infantilism.

4. Finally, genetic factors may indirectly alter the end organ through the nervous system, indicated by pathway A–C–D. Fibrocystic disease of the pancreas may be an example of this type of growth aberration.

From the previous facts it becomes apparent that whenever the medical practitioner is faced with a problem of marked deviation from the normal growth pattern, he must determine whether the deviation is due to: 1. one of the numerous extrinsic factors which may result in abnormal growth;

2. a congenital anomaly; 3. a mild constitutional difference in the endocrine system (in this type, the patient will show only minor deviation from the normal and the ultimate outlook is good); or 4. there is a serious disorder in the endocrine or neural systems. A complicating feature is that combinations of defects may occur, e.g., the syndrome of ovarian agenesis with dwarfism.

The hormonal influences on growth and sexual development are not, as yet, entirely understood. Figure 4 is a diagrammatic representation of the neuro-endocrine system as it relates to growth. For practical purposes it has been simplified and is guilty of numerous omissions. In general, debatable points are not included. The facts that the luteotrophic hormone may stimulate the adrenal cortex, and that the seminiferous tubules in the male under the influence of the lutienizing hormone may secrete a substance similar to progestin in the female are indicated by question marks.

There are six important trophic hormones elaborated by the anterior pituitary gland which play an important role in the growth pattern. All six are protein substances and all have been isolated in pure form with the exception of the follicular-stimulating hormone. These trophins activate the endocrine glands to produce hormones essential to normal growth. The circulating level of endocrine hormones causes the pituitary to produce more or less of the respective trophin and, at times, may cause an elaboration of more or less of a different trophin, e.g., an increased level of thyroglobulin may decrease the production of adrenocorticotropic hormone; or an increase in the level of circulating estrogen not only decreases the production of follicular-stimulating hormone but also increases the excretion of luteinizing hormone (the menstrual cycle). Basically, the six trophic substances act in the following manner:

1. *The Growth Hormone:* This is the only substance elaborated by the anterior pituitary which acts directly on the end organ to stimulate growth and development. It may also be important in carbohydrate metabolism and protein anabolism.

2. *The Thyrotrophic Hormone (TH)*: This tropin causes hyperplasia of the thyroid gland and a loss of colloids. Thyroglobulin is thus released to act on body metabolism.

3. *The Adrenocorticotrophic Hormone (ACTH)*: This protein acts directly on the adrenal cortex and stimulates the production of three classes of adrenocortins: corticosterones which cause gluconeogenesis replenishing liver glycogen storage; desoxycorticosterones which cause sodium and water retention and potassium loss; and androgens which cause skeletal and muscular maturation and the development of acne and secondary male sex characteristics.

4. *The Follicular Stimulating Hormone (FSH)*: In the ovary this hormone

is responsible for the production of the Graffian follicle which in turn elaborates estrogen and influences the development of secondary female sex characteristics. In the testis, FSH along with the luteinizing hormone causes the production of spermatozoa.

5. *The Luteinizing Hormone (LH):* This produces ovulation in the female and the formation of the corpus luteum. In the male, the luteinizing hormone is synonymous with the interstitial cell stimulating hormone. It causes the interstitial cells of the testes (Leydig cells) to produce androgen.

6. *The Luteotrophic Hormone (LTH):* This is the same substance as the lactogenic hormone or prolactin. In the non-pregnant female, this hormone maintains the corpus luteum for about two weeks and causes production of progestin which, in turn, influences endometrial changes. During pregnancy, LTH maintains lactation.

It is interesting to note that in the female, estrogen production is influenced primarily by the follicular stimulating hormone (FSH), while in the male, androgen production is controlled primarily by the luteinizing hormone (LH).

As indicated previously, the excretion of trophins by the anterior pituitary is influenced by the concentration of endocrine secretions in the circulation. In addition, anterior pituitary function is regulated by neural impulses originating in the hypothalamus. Therefore, disturbances in the hypothalamic region, e.g., craniopharyngioma, tumors, or hemorrhage can affect the growth pattern by altering the anterior pituitary control of endocrine secretion.

The practical application of the fundamental principles of hormonal influences on the growth pattern is the realization that disturbances may result from lesions in any of the three areas of control: the endocrine glands, anterior pituitary gland, or hypothalamus. The same symptom-complex may be produced by lesions in any one of these three locations, e.g., Cushing's syndrome may be the result of an adrenal tumor, basophilic adenoma of the pituitary, or possibly a hypothalamic lesion; sexual precocity in the female may be caused by an ovarian or adrenal tumor, a pituitary lesion or dysfunction of the hypothalamus; sexual infantilism in the male may be produced by lesions of the adrenal, anterior pituitary or hypothalamus.

A dwarf is any individual in whom a marked deviation from the normal growth pattern has resulted in stunting of growth. Table 1 lists the more important causes of dwarfism.

1. *Bone Diseases:* Chrondroplasia and dysostosis multiplex produce characteristic clinical syndromes. In almost all instances, the diagnosis is evident from clinical examination and x-ray changes in the osseous system. The other disorders in this group afford little difficulty in differential diagnosis.

2. *Nutritional and Metabolic Disturbances:* Celiac disease and fibrocystic

disease of the pancreas may retard the growth processes. An examination of the stool for its fat and starch contents, and an analysis of the duodenal juice for amylase and tryptin activity will establish the diagnosis. Renal insufficiency may be either glomerular or tubular. In either event, large amounts of calcium are lost in the urine which ultimately results in renal osteodystrophy. Chronic hepatic and pancreatic dysfunction may produce dwarfism.

TABLE 1

Causes of Dwarfism

I. *Bone Diseases*
- A. Chondrodystrophy and dysostosis multiplex
- B. Rickets, all types
- C. Osteogenesis imperfecta
- D. Disease of the spine, such as tbc. caries

II. *Nutritional or Metabolic Disorders*
- A. Celiac disease and cystic fibrosis of pancreas
- B. Chronic renal disease
- C. Hepatic insufficiency
- D. Nutritional defects and chronic infections

III. *Circulatory Disorders with Anoxemia*
- A. Congenital malformations of the heart
- B. Extensive chronic pulmonary disease

IV. *Endocrine Disturbances*
- A. Hypothyroidism
- B. Sexual precocity with early epiphysial fusion
- C. Hypopituitarism
- D. Mixed types

V. *Delayed Adolescence with Retarded Growth Spurt*

VI. *Primordial or Genetic Dwarfism (Constitutional)*
- A. Familial
- B. Sporadic
- C. Syndrome of ovarian agenesis and dwarfism

VII. *Unclassified Types*
- A. Dwarfism with progeria
- B. Dwarfism with severe brain defects

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3. *Circulatory Disorders and Chronic Pulmonary Disease:* The anoxemia caused by these conditions will retard the normal growth pattern. These are usually attended by cyanosis and clubbing of the fingers.

4. *Endocrine Disturbances:* After excluding the preceding entities, endocrine imbalance may then be considered as a possible cause of dwarfism. It is often difficult to differentiate the various types of endocrine imbalances, particularly before the age of twelve years. Hypothyroidism in childhood results in cretinism. The diagnosis is not difficult to establish. The marked

retardation in development of ossification centers, the characteristic facies, sluggish circulation, low basal metabolic rate, and elevated serum cholesterol together with a good response to thyroid replacement therapy adequately establish the presence of thyroid hypofunction. The hypopituitary or Loraine-Levi type of dwarf is symmetrically small. The development of ossification centers may be moderately or markedly retarded. After the age of twelve, the daily excretion of 17 ketosteroids will be less than 2 milligrams, and the total daily FSH excretion will not exceed 6 mouse units. These patients have a tendency to hypoglycemic attacks, and are particularly sensitive to insulin. Thorn has suggested that a response of the circulating eosinophils to ACTH together with a failure to respond to adrenalin may aid in differentiating hypopituitarism from other endocrine imbalances.

5. *Delayed Adolescence:* This is, perhaps, the most common cause of stunted growth. These patients are two or three years behind in osseous growth and in the development of ossification centers. Adolescence is delayed until sixteen or seventeen years of age. Then there usually occurs a rapid growth spurt with development of secondary sex characteristics or growth proceeds slowly until normal maturation is attained. Endocrine studies reveal no abnormalities. While these individuals remain small in stature, they ultimately reach the normal limits of growth and development.

6. *Primordial or Genetic Dwarfism:* There is no retardation of developmental processes or other evidence of endocrine disturbance. This type of dwarfism is an inherited trait and is a tribal characteristic of certain aboriginal pygmies. It may be transmitted through normal as well as dwarfed members of a family. A special type of genetic dwarfism is seen in the syndrome of ovarian agenesis and stunted growth. This group has premature development of ossification centers and early fusion of the epiphyses. They show an increase in excretion of FSH. The associated anomalies of webbed neck, coarctation of the aorta, and extraocular muscle weakness make the diagnosis a simplified one.

7. *Miscellaneous:* In this group progeria with dwarfism or the Hutchinson-Guilford syndrome is a rather rare entity characterized by premature fusion of the epiphyses, loss of hair and fingernails, sunken eyes, pinched nose and birdlike features, and joint deformities. Dwarfism also may be associated with severe brain defects.

The case presented represents a problem of anormal growth in which marked dwarfism has been produced. After an extensive laboratory work-up, two positive laboratory tests have more or less pin-pointed the site of pathology:

1. Hans Selye has termed the normal reaction of an individual to stress, infection, or trauma as "the adaptation syndrome." Under conditions of stress, the normal adrenal medulla secretes epinephrine and nor-

epinephrine. These substances ready the individual for any emergency by increasing cardiac output, elevating the blood pressure, and mobilizing liver glycogen. This is the first phase of "the adaptation syndrome." The epinephrine by way of the hypothalamus causes the anterior pituitary gland to excrete adrenocorticotropic hormone (ACTH). The ACTH, in turn, in the presence of an intact adrenal cortex, stimulates the production of adrenal cortins. The adrenal cortins replenish liver glycogen stores, and cause fluid and sodium retention. This is the second phase of "the adaptation syndrome." The carbohydrate regulatory hormones of the adrenal cortex (corticosterones), under the influence of ACTH, cause gluconeogenesis. In addition, they produce lysis of lymphoid tissue, and a fall in the circulating eosinophils. The most simple measure, then, of intact adrenal cortical function is supplied by the number of circulating eosinophils, i.e., the Thorn test. When a calculated dose of ACTH is administered to an individual with a normal adrenal cortex, it will cause a rapid drop in the circulating eosinophils. After the intramuscular administration of 5 milligrams of ACTH, the eosinophil level in this patient fell to zero within 3 hours. This indicates a normally functioning adrenal cortex. Again, when epinephrine is administered to a normal individual, it will cause a fall in the circulating eosinophils, but its mechanism of action is by way of the hypothalamus and anterior pituitary. A drop of 50 per cent or more in the circulating eosinophils is considered normal. When 3 minims of epinephrine were given subcutaneously to this infant, there was no fall in circulating eosinophils within 3 hours. Indeed, the number of eosinophils continued to rise. This fact together with the normal response to ACTH, indicates that the patient has a lesion in the hypothalamic-pituitary area.

2. The insulin sensitivity test is performed by injecting 0.1 unit per kilogram of body weight of regular insulin intravenously into a fasting patient. A fasting blood sugar level is determined and blood sugar determinations made at 15, 30, 60, 90, and 120 minutes respectively. In the normal individual, the blood sugar will fall rapidly within one-half hour and rise again to the fasting level between 60 and 90 minutes. In the presence of adrenal or pituitary insufficiency, a prolonged hypoglycemia will result. In this patient, 4 units of regular insulin were injected intravenously. At the end of 120 minutes, the blood sugar had not yet risen to the fasting level. This result indicates either adrenal or pituitary dysfunction. The normal Thorn test together with the absence of symptoms of adrenal insufficiency would again point to the pituitary as the site of pathology.

The exact nature of the basic pathological defect in this case cannot be stated with certitude. However, a logical sequence of events may be presumed. At birth, the patient weighed about 1.3 kilograms. The incidence of bleeding tendencies and permanent mental retardation in premature infants

of this size is exceedingly high. It is possible that this patient had an intracranial hemorrhage at birth which involved the hypothalamic-pituitary region. The air studies suggest focal cortical atrophy and would seem to corroborate this presumption. A hemorrhage into the site as described could account for all of the features in this case. The future of this infant is uncertain. However it is planned to institute a course of therapy with methyl testosterone and estrogen.

SUMMARY

1. A case of dwarfism occurring in one of twin infants and probably due to hypopituitarism has been presented.

2. The factors which affect the normal growth pattern, with particular emphasis on endocrine control have been briefly discussed.

3. A differential diagnosis of dwarfism has been attempted.

4. The abnormal laboratory findings in the protocol and their possible significance have been presented.

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NON-PENETRATING INJURIES TO THE ABDOMEN IN CHILDHOOD

Special Report No. 241

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INTRODUCTION

During the period from June 1935 to June 1952, there were 19 patients who suffered abdominal injury of an extent sufficient to require surgical intervention and repair. An analysis of these cases forms the basis for this report. This discussion will be confined entirely to non-penetrating wounds of the abdomen (17 cases) because they are more common than penetrating wounds and present greater difficulty in diagnosis and management. In non-penetrating wounds, there is more disagreement as to the necessity for operative intervention and as to the optimum time for surgery if it is deemed advisable. Accidental non-penetrating injuries to the abdomen are frequently sustained by children and the pediatrician often is called upon to render a decision as to the immediate disposition of the patient. Several

general principles must be kept in mind when dealing with a problem of this type, and they are as follows:

1. Any abdominal injury is serious and any person with abdominal trauma due to blunt force, even though first appearing slight, should be hospitalized since the clinical impression differs with the stage of the injury.
2. Early, a ruptured viscus may produce few signs and delayed rupture and late sequellae are relatively common.
3. The type of injury which will produce a non-penetrating abdominal catastrophe is also often the type which will cause concomitant damage in the form of multiple injuries. Head injuries, fractures of the ribs, and/or long bones, external blood loss and damage to the thoracic viscera are all conditions which not only complicate the clinical picture, but materially influence the subsequent course of treatment. For example, the patient with a head injury may be comatose and accurate evaluation of abdominal trauma is impossible. Injuries to the ribs may produce lung injury with hemo and/or pneumothorax which must be controlled before the patient is a candidate for abominal surgery.
4. There are certain accidental circumstances in childhood which are often associated with intra-abdominal injury; thus, when these situations are elicited in the history, the practitioner should be alert to the possibilities. These situations most often take the following form:
   A. Bicycle accidents, including abdominal impact against the handlebars (25 per cent of cases).
   B. Sledding accidents (10 per cent of cases).
   C. Falls from various heights and structures, such as from trees, fences, second story windows and the like (40 per cent of cases).
   D. Direct blows to the abdomen with violent force, such as a kick from a horse, running into a door knob, or having a hatrack fall across a child playing on the floor (15 per cent of cases).
   E. Automobile accidents. It should be noted that the leading cause of death between the ages of 1 and 4 years is accidents, and, of these, automobile accidents are the most common. Only 10 per cent of the patients in this series gave a history of automobile accident in association with their abdominal injuries. This low incidence might be explained by the fact that the average age of our patients was 10 years, which is a time when children are more subject to types of injuries other than from automobiles.

DIAGNOSIS

For clarity of thinking it is helpful to divide the intra-abdominal organs which can be injured into three main groups.

1. *Solid Viscera.* Included in this group are the liver, kidneys, spleen, and pancreas. In injury to the liver, the most immediate hazard is hemorrhage. Of secondary consideration is the bile peritonitis which later ensues. Statistically, the liver is the most commonly injured intraperitoneal organ. In our small series, injuries to the liver and kidney occurred with equal frequency (24 per cent). The liver is especially vulnerable because it is large, fixed, and fragile. Seventy-five per cent of our patients sustaining ruptured livers went into shock within 6 hours of the original injury and the average amount of blood found in the peritoneal cavity was 400 milliliters. The only postoperative complication encountered was the development of a subphrenic abscess in one instance.

The kidney was injured in about 29 per cent of our cases. In most instances this type of injury does not constitute an emergency as acute as the others, but may become so if excessive retroperitoneal hemorrhage, increasing shock, and peritoneal bulging occur. In this series, hematuria, gross or microscopic, was a constant finding. In each instance, a flat plate of the abdomen and intravenous pyelography was suggestive of injury to the right kidney which was confirmed at operation.

The spleen is a notorious cause of massive hemorrhage following abdominal injury. However, this form of injury was not encountered in the present series. Delayed bleeding does occur and the most constant signs are localized pain in the upper quadrant, often radiating to the shoulder with occasionally a palpable mass and dullness.

The pancreas is, perhaps, the least often injured to a serious degree and does not usually constitute the emergency from hemorrhage as does injury to the liver or spleen.

2. *Hollow Viscera.* This group is composed of the stomach, small and large intestines in addition to the gall bladder and urinary bladder. In general, perforation of the upper gastrointestinal tract produces an immediate chemical peritonitis perforation of the lower portion—a bacterial inflammatory peritonitis.

Rupture of a normal stomach is rare. Protection results from rib arrangement and the fact that it is not normally distended.

However, when a blow is received unexpectedly, the muscles of the abdominal wall are not contracted. This lack of muscle splinting leaves the viscera unprotected and exposed to the full power of an assailing force. When the small intestine becomes the victim of blunt trauma, the fixed segments are the areas most often involved; namely, A) the duodenum, B) near the ligament of Treitz, C) near the ileocecal region.

The small intestine was the site of rupture in 24 per cent of our patients and in each, the jejunum suffered lacerations an average of 4 inches from the ligament of Treitz.

When blows to the epigastrium injure the duodenum, the third portion is most frequently affected. This occurs because it is attached to the bodies of the upper lumbar vertebrae by peritoneum. A sudden striking force directed toward the epigastrium will drive the posterior wall of the duodenum against the surface of the vertebral bodies producing an injury to the individual wall. The bleeding, which may occur, has been known to dissect into the retroperitoneal spaces.

The same general factors produce rents in the vicinity of the ligament of Treitz. A potent force striking a segment of jejunum near this ligament moves the loop beyond its limit of motility, thus producing a tear. Lacerations of the ileum 2 or 3 feet proximal to the ileocecal valve result when the external force compresses the ileum against the vertebral column or pelvic bones. Another contributing factor may be the increase on intra-abdominal pressure from external violence.

Immediate and almost continuous vomiting with or without hematemesis is a constant finding in perforations of the gastro-intestinal tract.

It should be mentioned here that injury to the urinary bladder can occur without any direct blow to the abdomen. Comparison can be made to a balloon filled with water supported on a stick. Its momentum, in the event of a sudden stop, may result in its being torn away from its supporting structures, which in the case of the bladder, is the site of the prostatic urethra.

3. *Vascular Supporting Structures.* Damage here was to the mesentery of the small bowel in 5 per cent of our cases, and to the mesentery of the large bowel in 12 per cent.

The clinical picture presented by a patient with an injury to one or more of the structures mentioned will vary both with the severity of injury and the time at which the patient is first seen. Shock, of either neurogenic or vascular origin, was seen in 40 per cent of our patients within the first three hours after the injury. Twenty-five per cent of our patients passed this "stage of illusion" (varying from 1 to 5 days) during which symptoms improved at first and later returned in an aggravated form, requiring hospitalization. Vomiting is generally absent, unless the lesion involves a hollow viscus in which the incidence was about 90 per cent.

Pain is nearly always present, in a greater or lesser degree. There is, however, no relationship between the severity of the pain and the seriousness of the abdominal injury. The same may be said of tenderness. The site of localized tenderness and spasm will usually be a guide as to the location and nature of the underlying lesion.

When signs of general peritonitis exist most likely the small bowel has been ruptured or a hemoperitoneum has developed from extensive liver lacerations. If there is sufficient blood or fluid in the perioneal cavity, a

bogginess can be detected in the cul de sac on rectal examination or pain can be referred to the shoulder when the patient is placed in Trendelenberg position, and shifting dullness can be elicited.

It is always to be borne in mind that thoracic-cage and vertebral injury often produce spasm of the abdominal wall. Moreover, one should make every effort to recognize that tenderness and splinting can be due to direct injury of the abdominal wall without injury of a viscus. This is the so-called "peritoneal shock" comprised of abdominal pain, vomiting and clinical shock following trauma to the abdomen. Ten per cent of the patients in this series fell into this group and only acute gastric distension was discovered at operation. There are times when the history of a fall or abdominal injury may be misleading. In the presence of an obviously acute surgical abdomen with a history of a fall, one must keep in mind other causes for this condition. So often in children, the onset of acute appendicitis, rheumatic fever, or mesenteric adenitis may occur coincidentally with an injury but the latter, more dramatic happening, is recalled and attention erroneously focused upon it.

MANAGEMENT

The treatment of shock must be attended to first. Heat, oxygen, intravenous fluids, and morphine are the mainstays of treatment. The passage of a Levine tube with suction is valuable for several reasons:

1. Diagnostic aid if blood is present in the stomach;
2. Reduces shock if gastric dilatation is present;
3. An aid in operative technique if the abdomen is opened.

Where feasible, the treatment of shock and diagnostic procedures should proceed concurrently. Helpful diagnostic techniques are:

1. Repeated and meticulous examinations of the abdomen, including hernial openings with special notation of tenderness, spasm, rebound tenderness, and masses.
2. Pulse and blood pressure every 15 minutes until stabilized.
3. Patients should be crossmatched for at least one pint of compatible whole blood as a routine procedure.
4. Examination should be made of a voided urine specimen when possible, otherwise, a catheterized one. Bladder rupture may be suspected when one cannot recover the same amount of saline as is injected through the indwelling catheter.
5. Rectal examination to determine the presence of: (a) blood in the lower gastrointestinal tract; (b) bulging in the cul de sac; (c) spicules of bone from pelvic fractures and crepitation of air in pelvic connective tissue.
6. Roentgenograms: (a) an intravenous pyelogram if there is suspected kidney damage or if hematuria is present to determine both the site of

injury and the status or presence of the kidney injury; (b) a chest film may at times be helpful; and (c) flat plate of the abdomen in erect and supine position and left lateral decubitus position.

One must look for: (a) pneumoperitoneum (as little as 4 cubic centimeters of free air will show); (b) the condition of the lumbar vertebrae; (c) the presence of pulmonary pathology (both visceral and parietal); (d) the clearness of the psoas shadows; and (e) a generalized abdominal haze indicative of free fluid in the peritoneal cavity.

7. Repeat hematocrit determination until there is definite evidence of continued improvement or until laparotomy is decided upon.

Once it has been determined that an intra-abdominal injury is present, preparation should be made for laparotomy at the earliest possible moment. The case for conservatism is weak. While it is true that nothing may be done once the abdomen is opened, the diagnosis is so often in doubt, delayed rupture of either hollow or solid viscera is so common, and multiple lesions are so frequent that one had best look and see rather than wait and see.

After entering the peritoneal cavity, any frank bleeding should be immediately controlled after which a thorough and more systematic exploration can be done. Liver injury is often difficult to manage due to the friability of the organ and the readiness with which sutures tend to pull out. Mattress sutures offer the best type of security in this repair. The mortality and morbidity in liver injury have decreased markedly since the widespread use of oxycel and gel foam in the wounds of this organ. If the latter are not available, a small piece of muscle or omental fat may be substituted. Packing with gauze is dangerous and should be avoided. Splenic injuries are treacherous and where there is any significant damage, splenectomy is the procedure of choice. Most non-penetrating wounds of the kidney are of the bursting type and often involve the major vascular pedicle. Hematuria and bulging of the flank are the most suggestive signs and one should always know the status of the opposite kidney and be prepared to do a nephrectomy. Injury to the colon may occur away from the site of the abdominal trauma. If the extent precludes primary repair, resection with primary anastomosis should be done along with exteriorization and/or proximal colostomy.

MORTALITY

Ideally, four to six hours after the injury is the most favorable time for operation. Delay is the most common reason for a fatal outcome. Prior to the antibiotic era, the mortality rate from intra-abdominal injury was in the neighborhood of 40 per cent. There were no deaths among the 17 patients reported in this series. Seventy-five per cent of the patients sought

hospitalization within six hours of the injury and the mean amount of time that lapsed before surgery was ten hours.

A few illustrative cases are presented to amplify the problems and principles mentioned above.

CASE NO. 1—*Laceration, liver followed by subdiaphragmatic abscess and visceration*

This ten-year old colored boy was admitted to the hospital one hour after being struck by a truck. Not unconscious. Examination on admission revealed fracture of right arm and leg. Examination of abdomen revealed generalized moderate pain, and tenderness in all quadrants, with generalized spasm and generalized rebound tendencies. Patient later went into shock. The next morning the abdomen became distended and there was tenderness in the right quadrant. Taken to operating room; laparotomy revealed a 4-inch rent posterior border of the right lobe of the liver which was packed with 2 strips soaked with fibrofoam. Following operation, the patient was given about 2,000 milliliters of blood to maintain pressure. The patient had a stormy postoperative course and with the aid of x-rays a subdiaphragmatic abscess was diagnosed. On the 12th postoperative day the patient was again taken to the operating room where enclosure of the viscerated wound was done and also a rib was resected and a subdiaphragmatic abscess was drained. The patient continued to have temperature elevation during the following two months and about three months after admission another attempted subdiaphragmatic drainage was made but no pus was obtained. Following this the patient gradually improved and was discharged after about five months of hospitalization.

CASE NO. 2—*Ruptured kidney following injury. Nephrectomy*

This eleven-year old white female was sledding and hit a tree. She walked home one block away and then began complaining of right lumbar pain and pain in right lower quadrant. She passed bloody urine. Examination of the abdomen revealed swelling in the right upper quadrant and definite tenderness of the entire right side of the abdomen and costal vertebral angle. Entire abdomen moderately tender but not distended. There was a definite drop in hemoglobin and an elevated pulse. X-rays revealed fracture of the right seventh rib posteriorly. Excretory urogram showed the left kidney to be normal. The major portion of the right kidney, pelvis and calices were displaced downward and there was one separate area of opaque material lying under the 12th rib which was greatly separated from the rest of the dye. This was indicative of ruptured kidney. Exploratory laparotomy revealed the right kidney to be torn into three sections. The kidney was removed. The patient had an uneventful recovery.

CASE NO. 3—*Ruptured jejunum following injury*

This six-year old white female was admitted because she fell from a bicycle and was struck in the abdomen with a handlebar on the day of admission. She had generalized abdominal rigidity and tenderness. Laparotomy revealed that approximately six inches from the ligament of Trietz a small hematoma on the wall of the jejunum was present. Approximately four inches from the Trietz ligament a laceration (transverse) on the mesenteric side of the intestine was found. This measured approximately 3 centimeters in length and penetrated the lumen of the jejunum. Lacerations were repaired with fine chromic catgut. Patient had an uneventful recovery.

CASE NO. 4—*Meso-sigmoid tear following injury*

This ten-year old patient jumped from a third story window in a fit of temper. The abdomen, upon examination, revealed rigidity throughout. It was non-tender; peristalsis was not heard. X-ray examination revealed fracture of the right fibia and left metatarsal fracture. The patient was taken to the operating room where laparotomy revealed a meso-sigmoid tear which was repaired. There was no hemorrhage, and the patient had an uneventful recovery.

CASE NO. 5—*Abdominal injury—perforation of jejunum—serosal tears of stomach*

An old-fashioned hat rack fell upon this five year old colored male's abdomen. He immediately began to complain of generalized abdominal pain which seemed to later localize in the umbilical region. The child went into a semi-comatose condition and vomited. Vomitus contained a large amount of blood. Examination on admission revealed rigidity in both upper quadrants. There was tenderness over the entire abdomen, perhaps more around the umbilicus. The patient voided and the urine was grossly clear. Laparotomy revealed a small amount of blood in the peritoneal cavity. There was a 2.5 centimeter tear in the serosa on the anterior wall of the stomach, and another tear 3 inches from the ligament of Trietz which almost severed the jejunum. There was also a retroperitoneal hematoma. The child was given transfusions and supportive care. The patient had an uneventful recovery and was discharged three weeks after admission.

CASE NO. 6—*Acute gastric dilatation following injury*

This six-year old white female was admitted because of a fall down the stairs. She then developed severe abdominal pain. Examination of the abdomen revealed rigidity and tenderness in the upper abdomen. No peristalsis was heard. Tenderness in the right quadrant was noted. Patient was thought to have a ruptured spleen and laparotomy revealed only acute gastric distension. Patient had an uneventful recovery.

CASE NO. 7—*Hematoma of cecum and ascending colon*

This ten-year old white male was admitted because he fell from a bicycle five days before admission and since that time had complained of pain in the right lower quadrant. The pain was more severe than ever on admission. Had vomited almost continuously since injury. Examination of the abdomen revealed tenderness in the right lower quadrant, spasticity in the lower quadrant on the right. Laparotomy revealed a hematoma of the cecum and ascending colon. The appendix was not removed, the abdomen was closed and the patient had an uneventful recovery.

SUMMARY

Some of the salient features in the diagnosis and management of non-penetrating wounds of the abdomen in children are considered. Relevant data from 17 proven cases are described and discussed. It is stressed that concomitant injuries may complicate the diagnosis and alter the management; therefore, it is mandatory to evaluate the patient completely. Three groups of intra-abdominal structures, viz., solid viscera, hollow viscera and supporting structures may be injured singly or collectively; and some es-

sential features in the management of each are discussed. Illustrative cases are presented.

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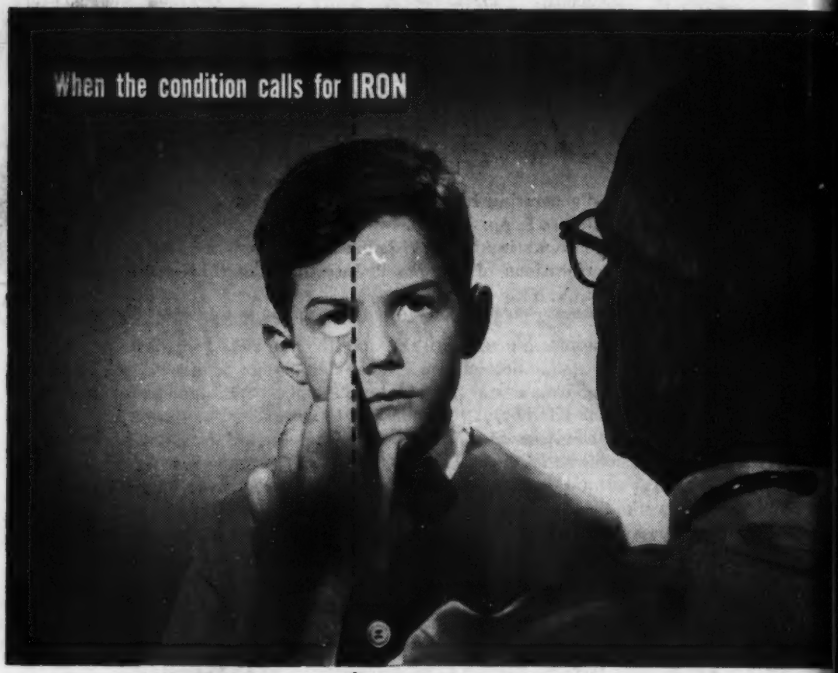

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